TYPHOID ILEAL PERFORATION

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY (BRANCH-I)

GENERAL SURGERY DEGREE EXAMINATION

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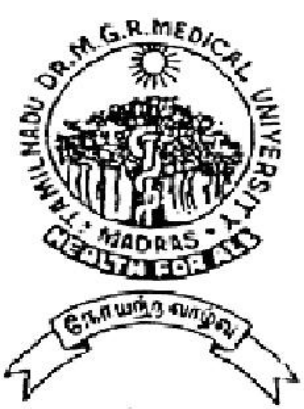

THE TAMILNADU

DR.M.G.R.MEDICAL UNIVERSITY

CHENNAI

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY OF TYPHOID ILEAL PERFORATION''** submitted by **Dr.Vinaykumar Gurumath** to the Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the requirement for the award of **M.S Degree Branch-1 (General Surgery)** is a bonafied research work were carried out under his supervision and guidance from 2009 to 2011.

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DECLARATION

I **Dr. Vinaykumar Gurumath** declare that, I carried out this work on, **"A STUDY OF TYPHOID ILEAL PERFORATION"** at the Department of Surgery, Government Rajaji Hospital during the period of 2009 to 2011. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any other university, Board either in India or abroad.

This is submitted to The Tamil Nadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

Place: Madurai Dr. Vinaykumar Gurumath

Date :

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CONTENTS

INTRODUCTION

Typhoid fever is endemic in South India especially seen in low socio-economic status. Typhoid fever is a protracted disease that includes bacteremic phase with fever and chills during the first week, wide spread reticuloendothelial involvement with rash abdominal pain and prostration in the second week and ulceration of Payer's patches with intestinal bleeding and perforation during third week.

It is one of the major cause of non traumatic ileal perforation in tropical countries like India. Ileal perforation is one of the most serious and life threatening complication of typhoid fever. Usually seen in second or third week after infection.

Perforation of ileum leads to contamination of peritoneal cavity with gastrointestinal secretions which may initiate catastrophic events which until stopped can be detrimental to the life of the patient. It is also challenging surgical emergency in

developing countries. Resulting peritonitis may be rapidly fatal unless it is treated promptly and vigorously.

Fifty patients were taken up for this study. In all cases perforation was established by clinical examination or radiology or laparotomy and typhoid fever was confirmed by blood culture or Widal reaction or histopathological examination.

Typhoid ileal perforations have high mortality and morbidity if not diagnosed properly. Conservative treatment is not suitable for ileal perforation. Surgery has been generally accepted as the treatment of choice. If patients are not diagnosed early, it progresses to death due to peritonitis. Mortality increases with delay in surgical intervention.

REVIEW OF LITERATURE

Whereas it was described as ‘Mesenteric fever’ by B AGLIVI in 1696, typhoid fever was given its universal name in 1834. The surgical treatment has been known since LEYDDEN (1884), L. EJARS (1903) and WEBBER, JOHNSON, OSLER (1917). Initially 100% death rate was reported for the perforations, now a days this mortality rate, although decreasing, still remains very high, ranging from 1 to 39%.

Kuruvilla in the year 1967, found a solitary perforation in the terminal ileum at a laparotomy done for suspected duodenal ulcer perforation. After taking the specimens at the edges of ulcer for histopathological examination lesion was closed. Widal test was positive and histopathological examination appearances were suggestive of typhoid ulcer.

In early sixties, Huckstep’s conservative management of typhoid ileal perforation became popular as result of higher morbidity and mortality associated with surgical intervention. With

improvement in surgical techniques & sepsis, pendulum has swung back in favor of surgical management of typhoid ileal perforation.

AIM OF THE STUDY

1. To study the age incidence and presentation of typhoid ileal perforation.
2. To study the outcomes in typhoid ileal perforation.

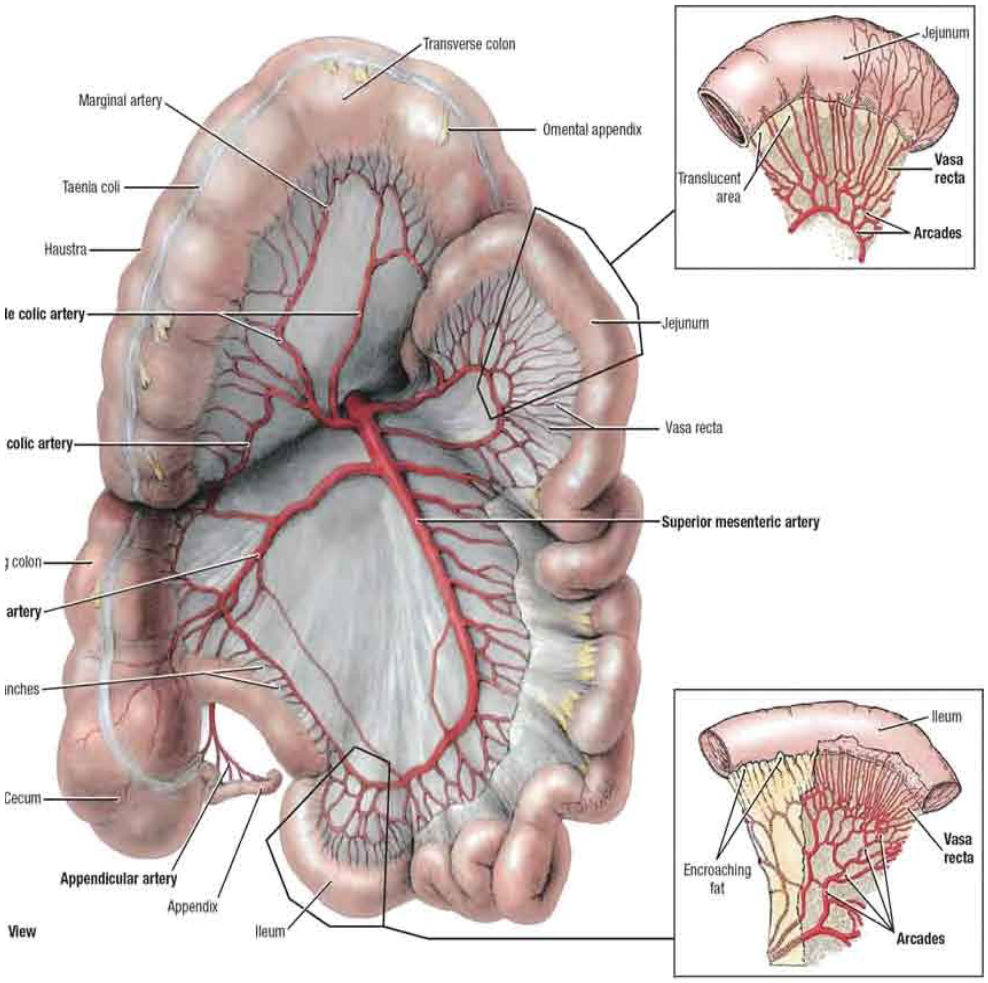
ANATOMY OF THE ILEUM

The ileum is the continuation of jejunum, measuring about 3-4 meters long, it is separated from caecum by ileocaecal valve. There is no line of demarcation between ileum and jejunum. However ileum is thinner than jejunum has more fat inside the mesentery than jejunum. Ileum has abundant Payer's patches unencapsulated lymphoid nodules. These form granular patches in the mucosa along the antimesenteric border.

Ileum is attached to the posterior abdominal wall by root of mesentery, which consist of two layers of peritoneum between which ileal branches and form a series of arterial arcades. Usually four to five arcades present, branches are accompanied by corresponding veins, nerve plexus and lymphatics.

Histologically ileum has 5 layers namely mucosa, submucosa, muscular layer, subserosa, serosa.

ANATOMY OF THE ILEUM

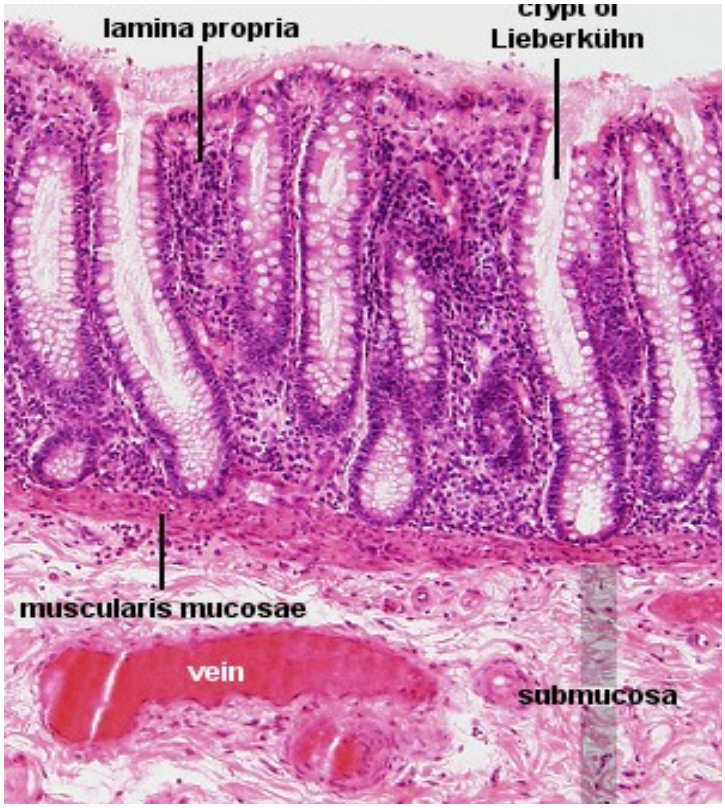


HISTOLOGY

Inner most layer is mucosa containing intestinal glands, villi and lymph follicles. Submucosa is made of loose areolar tissue in which blood vessels and nerves ramify in it. The muscle coat is having two layers viz, outer longitudinal layer and inner circular layer. The serosa of the ileum is the outermost layer formed by peritoneum, which covers the gut completely except along the mesenteric border.

There are two types of lymphatic tissue in ileum viz., solitary and aggregated. The solitary lymph follicles are scattered all over the mucosa, whereas aggregated lymph follicles occupy the antimesenteric border in a lengthwise manner. They are about thirty in number and are larger in the lower part of the ileum. They swell during enteric fever. Since they are in the antimesenteric border, the perforate the gut, contents of intestine will pass into the peritoneal cavity and set up peritonitis.

HISTOLOGY OF THE ILEUM



ETIOPATHOGENESIS AND PATHOLOGY

TYPHOID FEVER

Typhoid fever is an acute systemic illness caused by motile gram negative bacilli of the genus salmonella, usually S.typhi.

Humans are the only natural reservoir for S.typhi. Typhoid fever is acquired primarily through ingestion of contaminated water and food, much less commonly by direct finger to mouth contact with faeces, urine or other secretions. After ingestion organism invades the intestinal lymphatics and mesenteric nodes thus reach the blood stream. Once the bacteremia is established, it leads to the development of secondary areas of inflammation in the liver, gall bladder, spleen and bone marrow.

After one week of infection the bacteria are shed into the small bowel and appear in the stool.

The early change is hyperplasia of lymph follicles in ileum. The Payer's patches become swollen and ulcerated. This leads to capillary thrombosis and subsequent necrosis. In the second week necrosis and sloughing occur and ulceration of follicles leading on to perforation in the third week of disease.

PATHOLOGY

Salmonella invades intestinal epithelial cells as well as macrophages. Intra macrophage growth is important in pathogenicity and this seems to be mediated by bacterial genes.

Proliferation of macrophages swells the lymphatic submucosal nodules in intestine mainly in Payer’s patches of ileum. Later these Payer;s patches become prominent elevations in terminal ileum. In second or third week of infection the mucosa over the Payer’s patches shed leading to ulcer formation. Usually oval in shape with their long axis in longitudinal axis of ileum.

Once the infection is controlled these ulcers heal slowly with minimal scarring. Histologically mononuclear cells form nodular aggregates filled with red cells and nuclear debris. Polymorphonuclear cells are scarce found near the ulcerated surface of Payer’s patches. Lymphocytes and plasma cells found diffusely distributed.

In case of uncontrolled disease, there will be excessive proliferation of lymphoid tissue in Payer’s patches, resulting in rupture of necrotic patches leading to ileal perforations. Other

reticulo endothelial organs show marked histiocytosis mainly in spleen, liver, bonemarrow. Clinically hepatosplenomegaly is found. Microscopically reticuloendothelial organs show typhoid nodules with areas of necrosis.

DIAGNOSIS

SIGN AND SYMPTOMS:

The symptoms of typhoid ileal perforation are mainly abdominal pain with vomiting, associated with headache and fever. On examination there will be signs of abdominal tenderness which may be localized to right iliac fossa or diffuse with guarding and rigidity. It will be associated with abdominal distension, absent bowel sounds obliteration of liver dullness on percussion and free fluid in peritoneal cavity. Great emphasis has been given to above said sign and symptoms in making the diagnosis and deciding the criteria for inclusion of cases in our study.

In 1963, Franklin observed that most prominent features of typhoid ileal perforation are prolonged fever with diarrhoea and on examination diffuse abdominal pain with rebound tenderness in right iliac fossa.

History of prolonged fever with vomiting, diarrhoea with physical signs and symptoms suggestive of bowel perforation assume importance in making preoperative diagnosis of ileal

perforation. The laboratory tests like Widal reaction and blood culture are of less importance preoperatively.

WHITE CELL COUNT:

Typhoid fever is classically associated with decreased leucocyte count. But marked leucocytosis is seen in peritonitis secondary to any cause. So typhoid ileal perforations are usually associated with either high normal or leucocytosis.

WIDAL REACTION:

Widal reaction is based on identification of antibodies against Salmonella antigens. H-antigen(IgG) and O–antigen (IgM) agglutinating antibodies develop as early as fifth day after infection. These antibodies reach the maximum concentration during second or third week. Antibiotic administration in early period of infection leads to decreased antibody response resulting in negative Widal test. The rising titres of antibodies against S.typhi H & O Ag (more than 1 : 200 dilutions) in patient's serum is considered as positive test.

Kaul (1975) showed a positive widal test in 33% cases in their study, whereas Chauhan showed positive reaction in 70.1%

cases. Gandhi (1975) showed positive reaction in all his cases in study.

Saphala (1970) case study showed widal positivity in 80%. But Shah et al obtained negative reaction in 92% cases. Hence widal reaction is not a diagnostic test, it is to be taken as supportive for diagnosis.

BLOOD CULTURE:

Positive blood cultures seen mainly in first week of infection. As the typhoid ileal perforation usually occurs in second or third week of infection, blood culture will be usually negative. Moreover blood cultures are affected by prior antibiotic treatment in early period of infection. So blood cultures are less sensitive than Widal reaction in typhoid ileal perforation.

ABDOMINAL X-RAY:

Abdominal erect X-ray is taken to find out air under diaphragm (Pneumoperitoneum) in case of bowel perforation. The incidence of pneumoperitoneum is varying from one series to another.

ULTRASONOGRAPHY:

Ultrasonography abdomen shows mesenteric lymphadenopathy, free peritoneal fluid in peritoneal cavity with dilated bowel loops, sometimes omental adhesions in right iliac fossa.

CT SCAN ABDOMEN:

Contrast enhanced CT abdomen is useful in ileal perforation. It finds out mottled low density masses in the omental thickening of the bowel wall adjacent to mesentery, mesenteric lymphadenopaty, free fluid pelvis, hepatic pseudotumour appearance of typhoid nodules.

HISTOPATHOLOGICAL EXAMINATION:

Histopathological examination of the specimen taken from the edge of ulcer shows areas of necrosis surrounded by plasma cells, mononuclear cells, polymorphs containing abundant cytoplasm with bacteria and debris. These cells also called as typhoid cells. Evidence of typhoid inflammation was shown by

chronic non specific mononuclear infiltration in 30 cases from the biopsy of the ulcer edge in our study.

ETIOLOGICAL FACTORS

1. AGE INCIDENCE:

Typhoid perforation is seen in all the age groups. The age group ranges from 2 to 80 years in E.G. Archampong study(1976). According to Kuruvilla (1978), most of the perforations occur in the second and third decade of life, the age range was 8-57 years. Most perforations occurred in the age group 10-19 followed by 20-29 years. Similarly in Venkataramani Seetharam series the youngest patient was 13 years and the lowest was 65 years. 60.5% of their patients were in the second and third decades of life.

2. SEASONAL INCIDENCE:

Typhoid perforation occurs commonly in summer or autumn as seen in Kim et al (1975) study. Similarly in Swadia et al (1978) there was increased incidence of typhoid perforation in summer as well as in autumn.

3. GEOGRAPHICAL VARIATION:

Incidence of typhoid perforation varies in different parts of the world. High incidence of typhoid perforation is seen in West Africa. In Ghana successive studies over the past ten years, have

found a significant increase in the incidence of perforation from 15% reported by Badoe (1966) to 35% by Archampong (1974). Ikyrub et al (1972) obtained the phenomenal incidence of 33.6% from the same centre. The reason for increase in incidence is not known. Although typhoid infection is common in other parts of West Africa perforation is rarely seen.

4. SEX INCIDENCE:

Males are more commonly affected than females. The reason for the male preponderance is not known, may be related to increased risk of exposure to typhoid infection. In our study also male predominance is observed, out of 50 cases 33 were males. M.J. Kuruvilla showed the total of 31 patients with perforation of which 25 were male (9). In Venkataramani (1990) series there were 106 males out of 124 cases of perforation. Forrest et al obtained 62 male cases in 85 perforation cases. The male to female ratio is 2.4:1 in Kim series. 9:1 in Gandhi et al and 2.3:1 in Archampong series (1976).

5. SITE OF PERFORATION:

Typhoid perforation occurs predominantly in terminal ileum. Terminal ileum contains abundant Payer's patches which are predominantly infected in typhoid infection. Kim (1975) observed 80% cases of perforation occurred in the last 60 cm of ileum. Of which 72% perforations were within the last 40 cm. Similarly in Kuruvilla series (1978) perforations were confined to the last 30 cm of terminal ileum. Purohit et al noted all the perforations occurring within 40 cm from the ileocaecal junction.

TREATMENT

Typhoid ileal perforation is a surgical emergency. After vigorous resuscitation and correction of dehydration & electrolyte imbalance all cases should be planned for definitive surgical intervention. Depending on the extent of ileum involved and general condition of patient surgical procedure varies. In any case, active early intervention reduces morbidity and mortality.

In the early sixties, conservative management of typhoid ileal perforation was favored. In 1960, Huckteps proposed the non surgical management of typhoid ileal perforation which was supported by Norman Taylor. As the surgical techniques were improved operative approach started getting more importance, and decline in mortality rate was observed with early surgical interventions.

In 1963, Li Franklin raised the following objects against the rationale of non operative management – With adequate gastro intestinal suction and specific antibiotic therapy further perforation

is unlikely. Evacuation of pus from the peritoneal cavity reduces the toxemia and enhance the recovery of the patient.

MANAGEMENT OF PERFORATION

Patient's dehydration should be corrected. Preoperative antibiotic to cover both aerobic and anaerobic organism should be administered. Patients should be put on nasogastric aspiration and catheterised to monitor urinary output. Any imbalance in serum electrolyte concentration should be corrected accordingly. Depending on the general condition of patient and status of ileum, operative procedures differ.

SIMPLE CLOSURE OF PERFORATION

This is simple and standard procedure for ileal perforation. After thorough laparotomy perforation site is sought out. All toxic fluid will be let out. Perforation is closed with atraumatic needle in two layers using 3-0 vicryl for full thickness and 3-0 silk for seromuscular layers. After thorough peritoneal lavage, flank drain will be kept in pelvis and hepatorenal pouch. Abdomen closed in layers.

Archampong (1976) noted that the more insidious the perforation, the greater are the bowel inflammation and edema at the time of perforation, causing greater friability and increased difficulty in handling and suturing the bowel during surgery. In 1985, Chauhan et al treated 138 cases of typhoid perforation surgically. In these patients, the principal operation was closure of the perforation and peritoneal drainage. The overall mortality of surgical treatment was 58.7%. Solitary perforations were treated by simple closure in several patients of Kuruvilla et al 1978. Forrest et al (1981) treated forty three patients of typhoid perforation with simple closure.

Where there were only one or two perforations simple closure had the advantage of being quick and easy. After vigorous resuscitation, simple closure of the uncomplicated solitary perforation and peritoneal lavage will suffice for most of the cases. Of simple closure, double layer closure of perforation lowered the mortality rate in comparison with single layer closure.

In Swadis (1978) study of 112 cases, 104 cases were treated by simple closure with only two cases of developing post operative

leakage and fistula. Thus the incidence of leakage was quite low and possibility of' reperforation or leakage after simple closure has been unduly exaggerated. Hence uncomplicated solitary perforation without external evidence of other ulcers was treated b simple closure without undue tension.

The operative procedure depends on general condition of the patient and on surgeon's preference. But kim (1975) treated 103 cases with simple closure and had mortality of only 10% whereas Purohit et al (1976) treated ten patients of typhoid perforation by simple closure after trimming the edge and had cent percent success and claims it to be the ideal operation as it is quick and easy, satisfies requirement of wound healing, avoids inverting second layer stitches which often cut through the friable ileal wall. And also facilitates confirmation of diagnosis by biopsy of trimmed tissues. The average morality of about 25% in Typhoid ileal perforation treated by simple closure led various authors to device various

techniques to overcome the high mortality in an attempt to find out an ideal operation which would prevent re-perforation and other complications.

ALTERNATIVE OPERATIVE PROCEDURES:

- Resection of the affected segment with an end to end anastomosis.
- Simple closure after excision of seromuscular layer surrounding the perforation.

RESECTION OF THE AFFECTED SEGMENT WITH END TO END ANASTOMOSIS:

In typhoid ileal perforation, where there were multiple perforations or multiple ulcers or where ileal segment looked unhealthy or where perforation was accompanied by hemorrhage resection becomes the operation of choice. To start with, the veins were tied off and the segment intended for resection was clamped. Manipulation was kept to the minimum and care was taken to see that both sides of anastomotic site had adequate blood supply. Its advantages are:

1. Resection prevents re-perforation

2. It also prevents further perforation of nearby ulcer.

M.J. Kuruvilla (1978) treated 11 cases of their series with of anastomosis. Mulligan et al (1972) write ‘at operation, resection seems rationale on feature to do more than merely repair the perforation. Kim (1975) obtained lower mortality in his series treated by resection when compared to simple closure.

SIMPLE CLOSURE AFTER TRIMMING THE EDGES OF PERFORATION:

It consists of simple closure of perforation after refreshening the edges combined with or without oversewing areas of apparent impending perforation. Purohit treated 10 patients of perforated ileum by simple closure after trimming the edges and had cent percent success rate. In Forrest 1981 series, 43 patients were treated by simple closure after refreshening the edges combined with oversewing the areas of impending perforation. But their series showed 30.2% of mortality rate.

BLIND DRAINAGE:

It is nothing but bilateral flank drainage under local anesthesia without laparotomy which could not be performed

because of very poor general condition of the patient who could not tolerate the great surgical manoeuvre. Kuruvilla M.J. (1978) treated three cases with blind drainage because of poor general condition, of which two patients died in days after drainage.

Blind drainage may help those patients who are too ill to withstand laparotomy. In most of the series, blind drainage procedure claimed cent percent mortality rate. Kala et al (1978)

and Badoe. F. Series obtained 60% and 100% mortality rate respectively with blind drainage. Kim et al of Korea showed 20% of mortality rate in their study with blind drainage.

All cases of typhoid ileal perforation were given Inj. Ceftriaxone for 5 days followed by Tab. Ciprofloxacin for 2 weeks.

ANAESTHESIA FOR SURGICAL PROCEDURES OF ILEAL PERFORATION:

In most of the cases of typhoid perforation, spinal anesthesia is preferred for surgical treatment. Those with hypotension or those who needed only blind drainage because of poor general condition,

we can use local anesthesia. In few cases with otherwise good health, general anesthesia is used.

COMPLICATIONS

Various complications can occur following surgical treatment Viz.

EARLY COMPLICATIONS:

Toxaemia, sepsis, respiratory infections, paralytic ileus, Intra abdominal abscess, transfusion reaction, meningism, reperforation.

LATE COMPLICATIONS:

Wound infection, wound dehiscence, decubitus ulcer, burst abdomen, faecal fistula, Intestinal obstruction, incisional hernia.

Apart from the wound infection, the most common abdominal complication is wound dehiscence occurring in up to 30% (Shah—1967). This is the reflection of both the high incidence of infection and debility of the patient. As like as wound dehiscence, the development of faecal fistula is catastrophic. This may be the result of reperforation, perforation in another area or the

results of suture line break down. Its incidence varies from 4% to 21%. It has been reported in every type of surgery.

MATERIALS AND METHODS

Fifty patients with ileal perforation who underwent surgery were included in this study from the year 2009 to 2011. In all the fifty cases pneumoperitoneum was confirmed by either clinically or radiologically. Typhoid etiology was confirmed by blood culture or Widal test or histopathological examination.

In all cases, routine investigations like haemoglobin, blood urea, Random sugar, serum creatinine and electrolytes done. Plain X ray abdomen erect and chest x ray PA view taken. Intraoperatively biopsy was taken in all the cases to confirm the etiology. Finally in all cases history, examination findings, line of management were recorded and followed up postoperatively.

ILEAL PERFORATION AT THE ANTIMESENTERIC BORDER

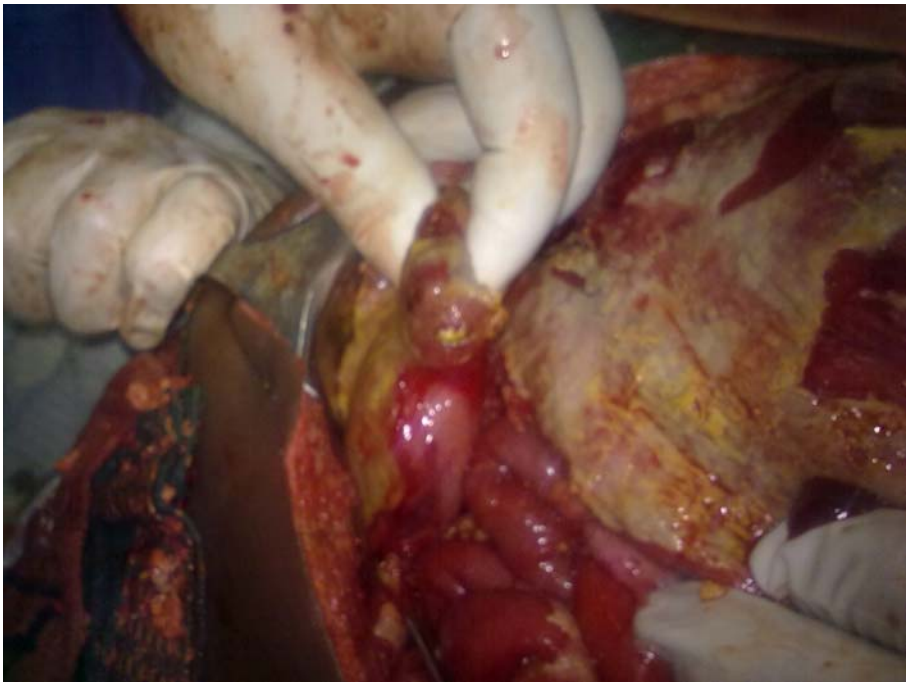
EXCLUSION CRITERIA:

Ileal perforations secondary to trauma, tuberculosis, volvulus, strangulated hernia, necrotizing enteritis, mesenteric ischaemia were excluded from our study.

Surgical procedures adopted:

- Simple closure of perforation done in 35 cases.
- Resection of affected segment of bowel and end to end anastomosis done in 11 cases.
- Blind flank drainage done in 4 cases.

ILEAL PERFORATION SITE EXPOSED



CLINICAL DATA

In our study, we included 50 cases of typhoid ileal perforation. Ileal perforations due to other causes like trauma, tuberculosis, volvulus, intussusception, strangulate hernia, mesenteric vascular occlusion were excluded. In all cases, history is noted. Clinical findings recorded, all cases were treated surgically, intraoperative findings recorded. All cases were followed up postoperatively for 6 months.

In our study of 50 patients, Typhoid ileal perforation has occurred in 33 males (66%) and 17 females (34%), youngest patient in our study was 15 years and oldest was 72 years. 76% of cases were in second to fourth decades of life span (Table-I)

Majority of cases presented with symptom complex of headache, fever abdominal pain, distension vomiting. On examination varying degree of abdominal tenderness, guarding, rigidity was found. Duration of each symptom is recorded (Table-II).

96% cases presented with abdominal pain whereas abdominal distension (66%), tenderness (80%) guarding (70%) in our study (Table-II).

The diagnosis was more difficult however in two categories of patient, a small group of patient in whom perforation occurred while under medical treatment and patients with protracted illness reaching several days after perforation. In the former group the signs of perforation were in abeyance because the complication developed insidiously. In the latter group gross abdominal distension over shadowed the other signs of perforation.

Widal Reaction:

Widal test was done in all cases in our study. Positive widal reaction was seen in 33 cases (66%) and negative in remaining cases (34%). The widal reaction was more often positive than blood cultures in our study (Table-V).

Blood culture:

Blood culture was done in all cases in our study. Positive blood culture for Salmonella typhi observed in only 16 cases (32%) (Table-V).

ILEAL PERFORATION SITE EXPOSED

Histopathological Examination:

In our study, intraoperatively biopsy was taken from the edge of ulcer and subjected for HPE. HPE suggestive of typhoid was seen in 30 cases (60%) (Table-V).

White Cell Count:

Normal leucocyte count was seen in 10 cases (20%). High normal leucocyte count (8000-11000/cumm) observed in 23 cases (46%). Leucopenia seen in 9 cases (18%) and leucocytosis seen in 8 cases (16%) (Table-III).

X ray abdomen Erect:

X ray abdomen erect was taken in all cases preoperatively. Pneumoperitoneum was seen in 70% cases.

Etiological Factors

AGE INCIDENCE:

In our study, typhoid ileal perforation is seen in second decade through seventh decade. Most of the perforations occurred in second and third decade (22% cases) (Table-I).

SEX INCIDENCE:

Typhoid ileal perforation in our study was seen in 66% males and 34% females (Table-I).

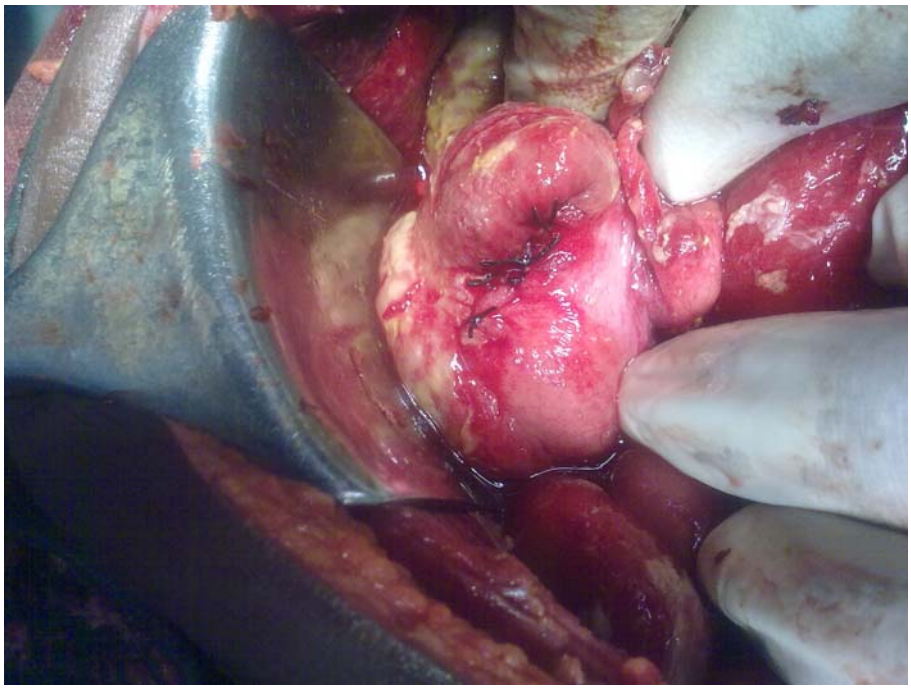
SITE OF PERFORATION:

In 29.63% cases, perforation was confined to terminal ileum, last 25 cm from ileocaecal junction. In 30% cases, perforation was located between 25 and 50 cm from ileocaecal junction (Table-VI).

FEVER-PERFORATION INTERVAL:

As seen in other studies, perforation occurred in early course of disease in our study. 45 cases (90%) presented with perforation in early course of disease (< 2 weeks) (Table-XI). 88.88% patients who perforate during the first two weeks, of illness appear to have a better prognosis in typhoid ileal perforation.

PRIMARY CLOSURE OF ILEAL PERFORATION



TREATMENT

Various surgical methods were adopted for the present series.

SIMPLE CLOSURE OF THE PERFORATION

Since the perforations were solitary one in 96% patients the principal operation was closure of the perforation and a flank drainage. Following simple closure 8 patient out of 35 perforation patients died, due to various complications showing the mortality rate of 22.85 % (Table - IX).

RESECTION AND END TO END ANASTOMOSIS

Resection of the affected segment of ileum was performed in cases where there were multiple perforation as well as multiple ulcers with 36.36% mortality (Table-IX).

BLIND DRAINAGE

Because of poor general condition, bilateral flank drainage was performed under local anesthesia in 4 cases who would not

tolerate great surgical manoeuvre. In this series, the mortality rate with blind drainage was 50% (Table - IX).

POST OPERATIVE COMPLICATIONS:

In the present series wound sepsis is the most common complication in 40% cases whereas the post-operative period was uneventful in 48% cases. Other complications like toxemia and paralytic ileus were developed in 32% and 20% cases respectively(Table-VII).

Our observations was similar to Forrest's view that wound dehiscence occurring in 6% cases, whereas in Forrest et al series, wound dehiscence was occurring in upto 6%.

The incidence of faecal fistula was 6.0% approaching to Swadia (1978) series. Its incidence was high upto 21% in Olurin series and Forrest series. The high fistula rate is the result of both poor wound healing and the nature of the lesion in the intestine.

Other complications included in this series are transfusion reaction Nil, Thrombophlebitis (2%), Pelvic abscess 4% and respiratory complications 14% (Table-VII). Only one case developed intestinal

obstruction (4%) six months after simple closure of perforation. That patient underwent laparotomy again and the adhesions were released.

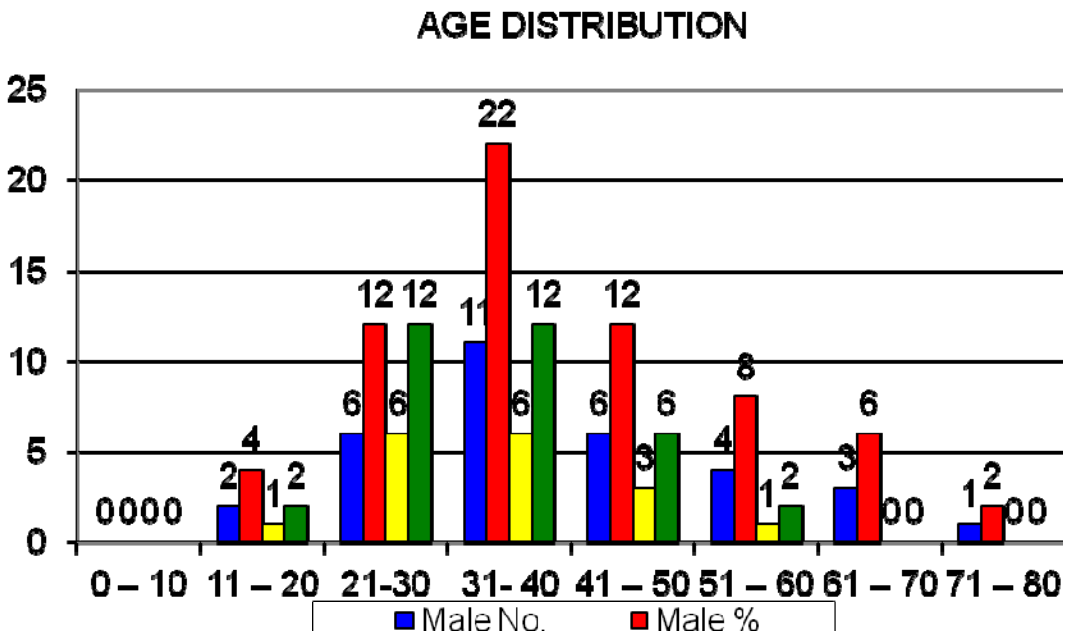
MORTALITY RATE:

The overall mortality rate of Typhoid perforation in the present series was 28%. It was 50% with blind drainage (Table-IX).

Table – I

AGE AND SEX DISTRIBUTION

Age Group	Male		Female		Total
	No.	%	No.	%	
0 – 10	0	0	0	0	0
11 – 20	2	4	1	2	3
21-30	6	12	6	12	12
31- 40	11	22	6	12	17
41 – 50	6	12	3	6	9
51 – 60	4	8	1	2	5
61 – 70	3	6	0	0	3
71 – 80	1	2	0	0	1
Total	33	66	17	34	50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6 12 6 12
11 22 6 12
6 12 3 6
4 8 1 2
3 6 00
1 2 00
0 – 10
11 – 20
21-30
31- 40
41 – 50
51 – 60
61 – 70
71 – 80
Male No.
Male %

Table – II

SIGNS AND SYMPTOMS

No.	Symptoms	Number of cases	Percentage
1.	Head ache	25	50
2.	Fever	42	84
3.	Vomiting	16	32
4.	Abdominal pain	48	96
5.	Bowel Habits		
	Normal	1	2
	Constipation	33	66
	Diarrhoea	12	24
6.	Distension	35	70
7.	Respiratory	6	12
8	Anuria	3	6
	SIGNS		
9.	Distension	33	66
10.	Abdomen Tenderness	40	80
11	Rigidity	30	60
12	Guarding	35	70
13	Free fluid	10	20
14	Bowel Sounds		
	Normal	4	8
	Absent	32	64
	Increased	3	6
15	Obliteration of Liver Dullness	34	68

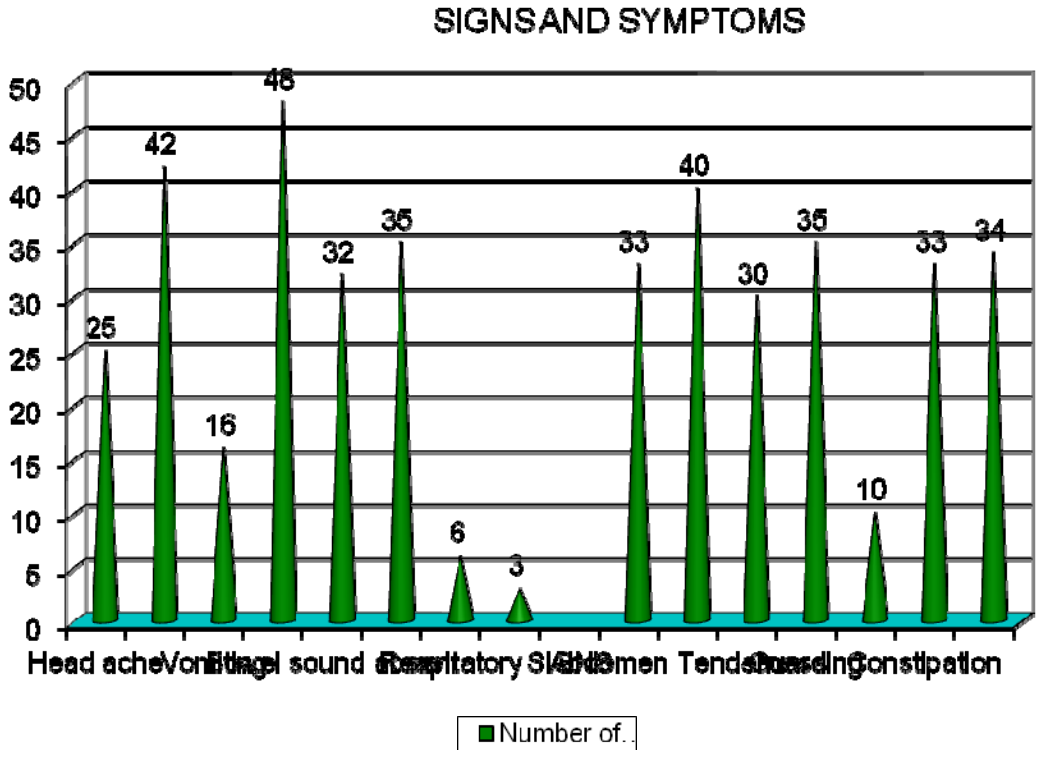
SIGNSAND SYMPTOMS
50
45
40
35
30
25
20
15
10
5
0
25
42
16
48
32
35
6
3
33
40
30
35
10
33
34
Head ache
Constipation
Number of..

Table – III

LABORATORY INVESTIGATIONS

SNo.	Laboratary Investigations	No.of patients	Percentage
1.	Leukopaenia < 4000 / cumm)	9	18
2.	Normal Total Leucocytes Count (4000 – 8000)	10	20
3.	High Normal Total Leucocytes (8000 – 11000 / cumm)	23	46
4.	Leucocytosis (>11000 / cumm)	8	16

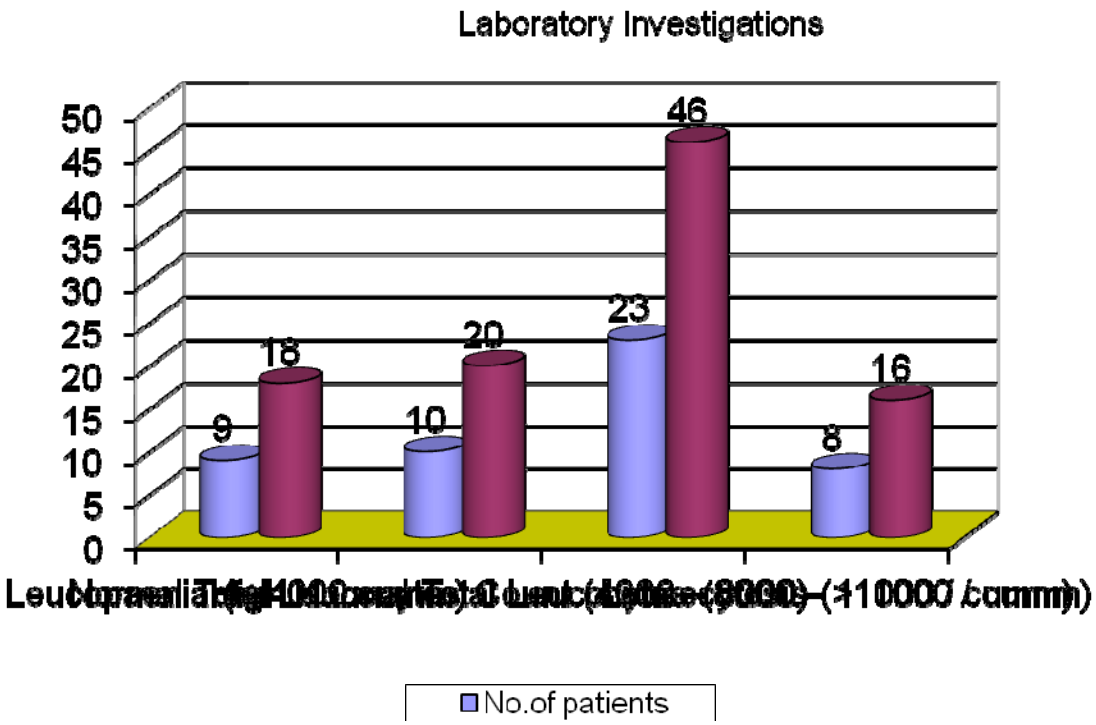
Laboratory Investigations
50
45
40
35
30
25
20
15
10
5
0
9
18
10
20
23
46
8
16
No.of patients

Table -IV

ELECTROLYTE CHANGES

S.No.	Serum Potassium ion level	No.of cases preoperative	No.of cases post operative	Deaths	% of mortality
1.	Normal level	24	36	6	16.67
2.	Low Level	26	14	8	57.14

Table – V

SEROLOGICAL TESTS AND HPE

S.No.	Name of the test	Positive Cases	Percentage	Negative cases	Total
1.	Widal Test	33	66	17	50
2.	Blood culture	16	32	34	50
3.	HPE	30	60	20	50

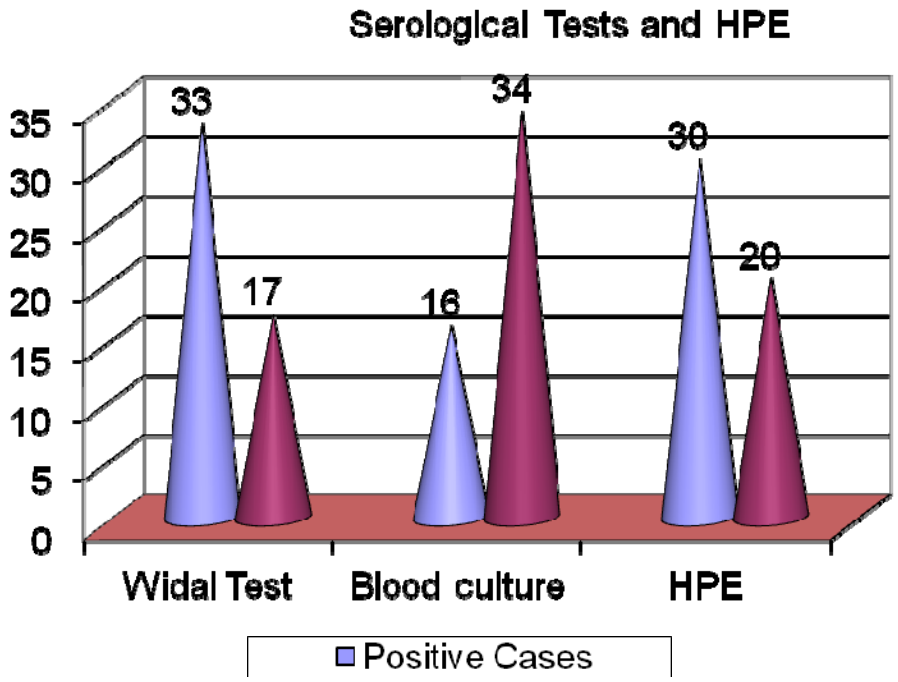
Serological Tests and HPE
35
30
25
20
15
10
5
0
33
17
16
34
30
20
Widal Test
Blood culture
HPE
Positive Cases

Table – VI

SITE AND NUMBER OF PEFORATIONS

Site of perforation from ileocaecal junction	No.of cases	Death	%
0 – 25 cm	27	8	29.63
25 – 50 cm	20	6	30%
> 50 cm	3	0	0

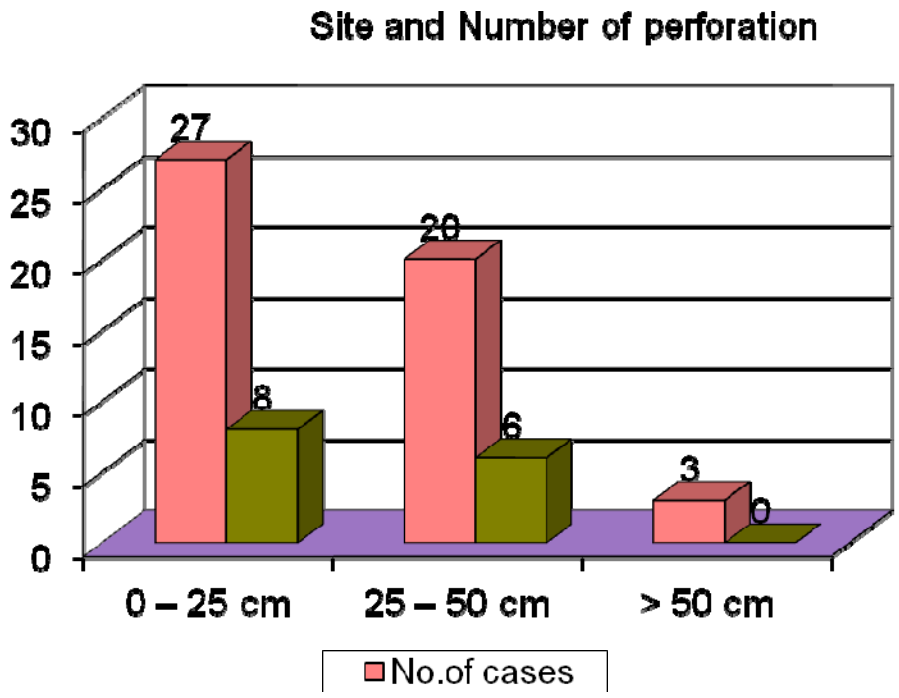
Site and Number of perforation
30
25
20
15
10
5
0
27
8
20
6
3
0
0 – 25 cm
25 – 50 cm
> 50 cm
No.of cases

Table – VII

COMPLICATIONS

No.	Complications	No of cases	%
1.	Wound dehiscence	3	6
2.	Toxemia	16	32
3.	Paralytic ileus	10	20
4.	Sepsis	20	40
5.	Transfusion reaction	0	0
6.	Respiratory	7	14
7.	Faecal fistula	3	6
8.	Pelvic abscess	2	4
9.	Thrombophlebitis	1	2
10.	No infection	24	48
11.	Encephalitis	0	0
12.	Late complication Intestinal obstruction	2	4

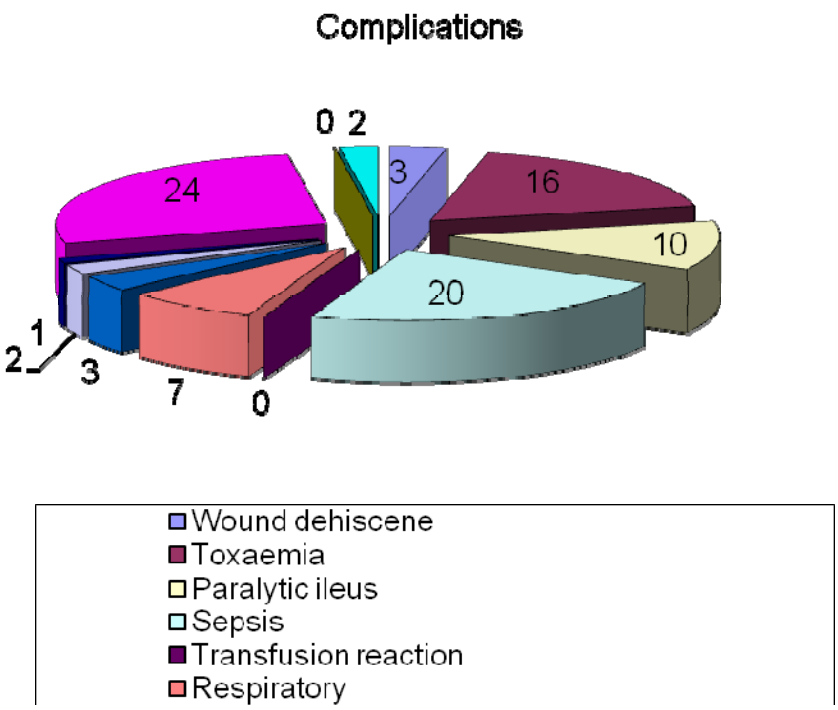
Complications
0
2
3
16
10
20
0
7
3
2
1
24
Wound dehiscene
Toxaemia
Paralytic ileus
Sepsis
Transfusion reaction
Respiratory

Table – VIII

CAUSE OF DEATH

S.No.	Causes of Death	No of cases	%
1.	Circulatory failure	2	14.29
2.	Respiratory complication	2	14.29
3.	Toxemia	9	64.29
4.	Faecal fistula	1	7.14

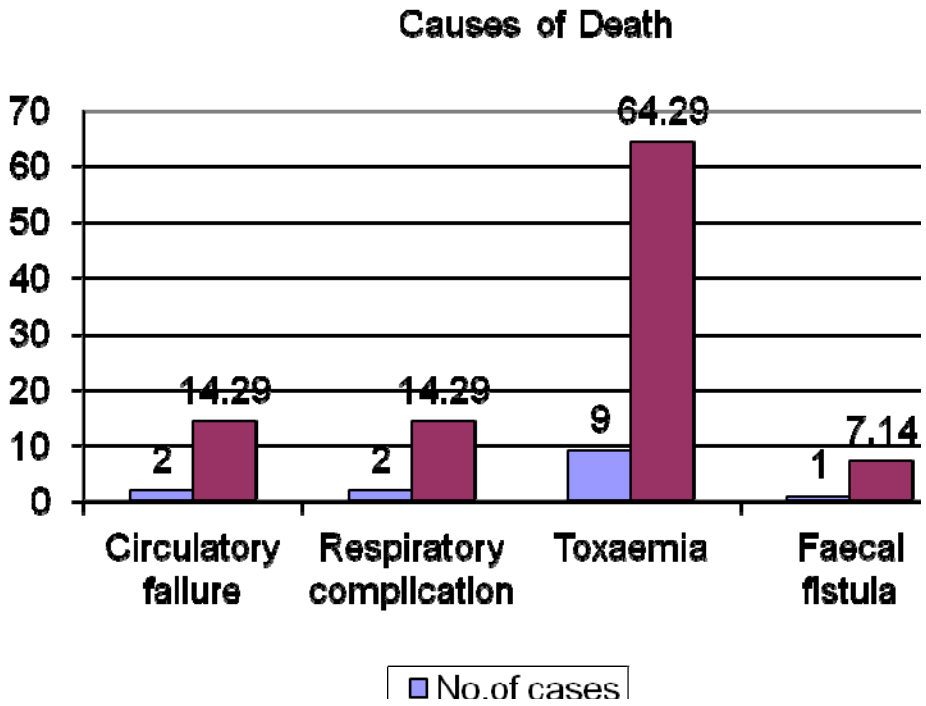
Causes of Death
70
60
50
40
30
20
10
0
64.29
14.29
14.29
9
7.14
2
2
1
Circulatory failure
Respiratory complication
Toxaemia
Faecal fistula
No.of cases

Table – IX

METHOD OF TREATMENT & MORTALITY

Method	Alive		Death	
	No.of cases	%	No.of cases	%
1. Simple closure	35	27	8	22.85
2. Resection a Anastamosis	11	7	4	36.36
3. Flank drain	4	2	2	50
Total	50	36	14	

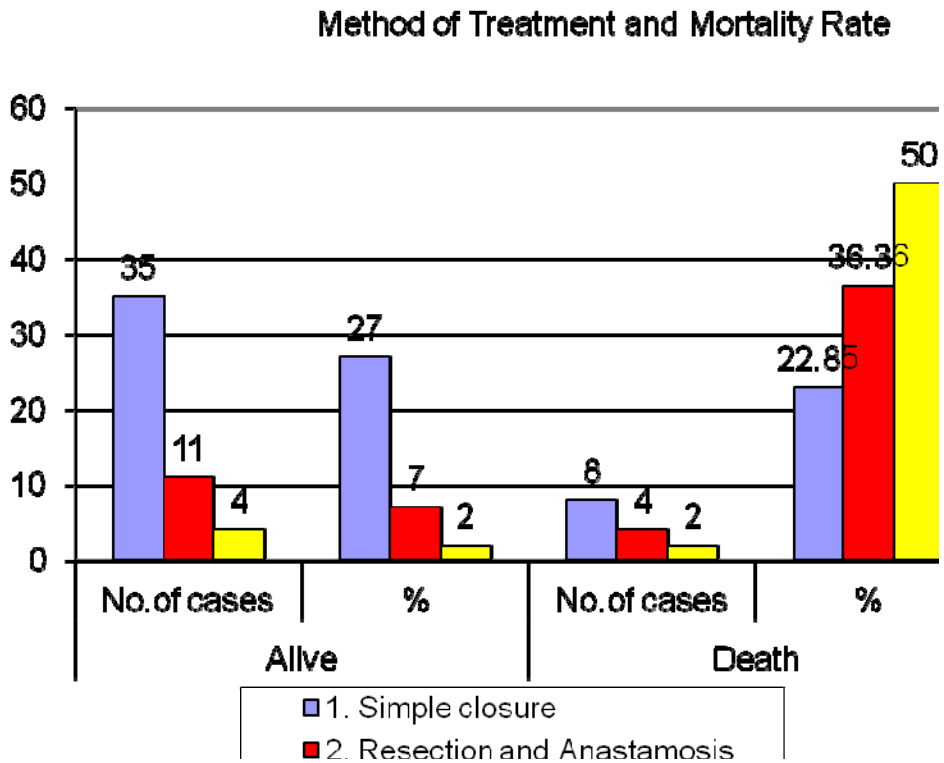
Method of Treatment and Mortality Rate
60
50
40
30
20
10
0
35
11
4
27
7
2
8
4
2
22.85
36.36
50
No.of cases
%
No.of cases
%
Alive
Death
1. Simple closure
2. Resection and Anastamosis

Table – X

PROGNOSIS OF SURGERY Vs CONSERVATIVE TREATMENT

Parameter	No of cases	Alive		Death	
		No.	%	No.	%
Surgery	46	34	73.91	12	26.09
Flank Drain	4	2	50	2	50

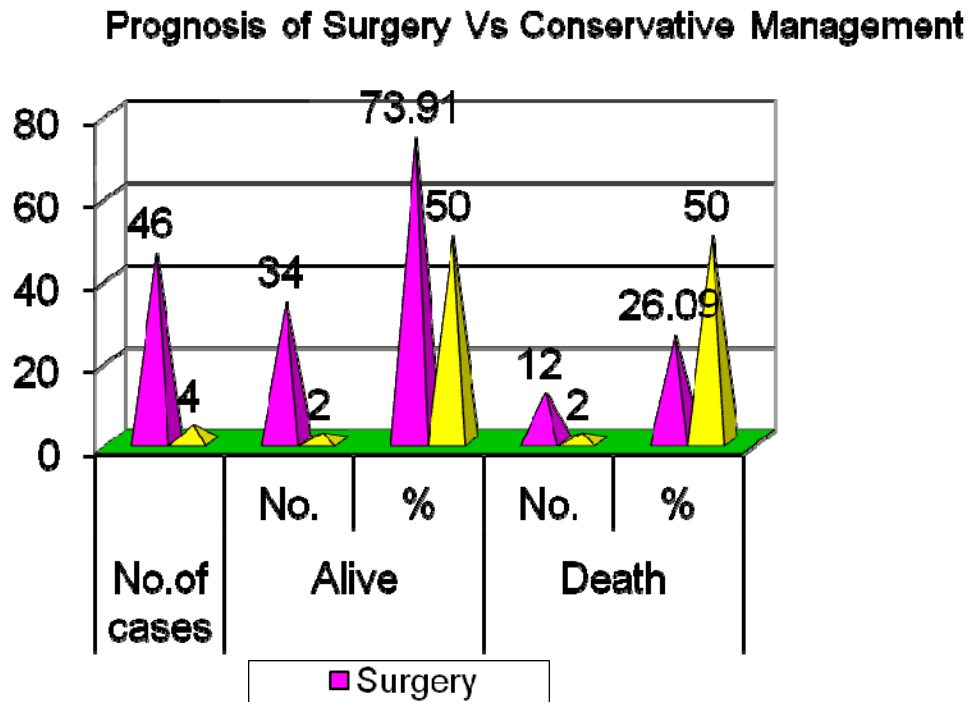
Prognosis of Surgery Vs Conservative Management
80
60
40
20
0
46
4
34
2
73.91
50
12
2
26.09
50
No.
%
No.
%
No.of cases
Alive
Death
Surgery

Table – XI

MORTALITY IN RELATION TO ONSET OF

PERFORATION & SURGERY INTERVAL

S.No	Perforation surgery interval	Alive	Deaths	Total	Percentage
1.	24 hrs	11	1	12	8.33
2.	24 – 48 hrs	10	2	12	16.67
3.	48 – 72 hrs	9	2	11	18.18
4.	72 – 96 hrs	4	4	8	50
5.	Above 96 hrs	2	5	7	71.43

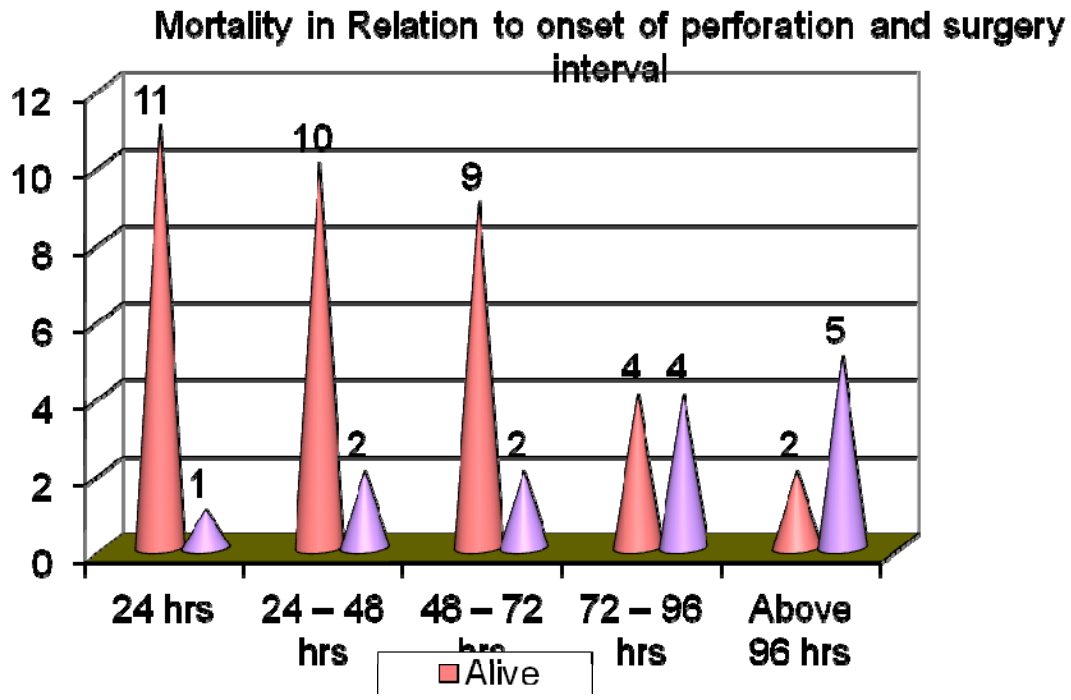
Mortality in Relation to onset of perforation and surgery interval
12
10
8
6
4
2
0
11
1
10
2
9
2
4
4
2
5
24 hrs
24 – 48 hrs
48 – 72 hrs
72 – 96 hrs
Above 96 hrs
Alive

Table – XII

PATTERN OF TYPHOID FEVER COMPLICATIONS

S.No	Duration	No.of cases	Deaths	% of mortality
1.	0-5 days	8	1	12.25
2.	6 – 12 days	27	7	25.93
3.	11-15 days	10	3	15
4.	> 2 weeks	50	14	28

Pattern of typhoid fever complication (Ileal perforation)

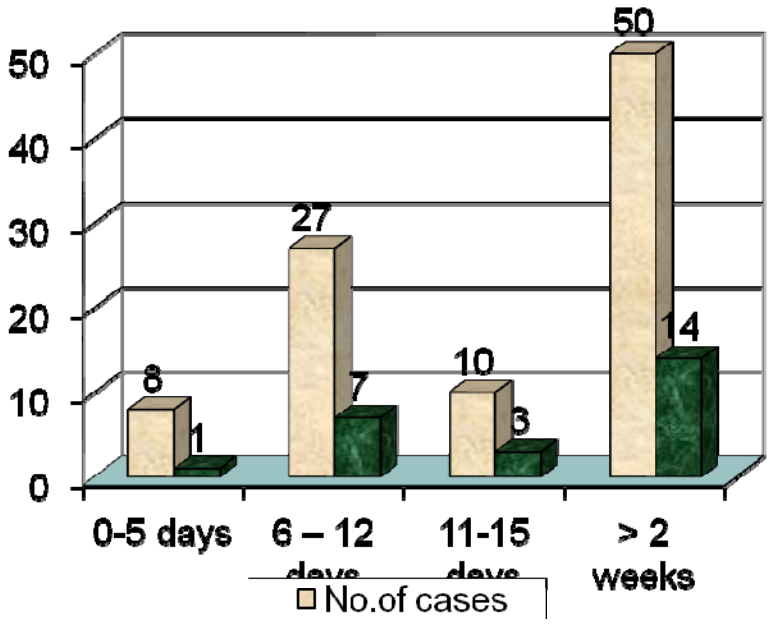

Table –XIII

NUMBER OF PERFORATIONS

	No.of cases	Death	%
Single	48	12	25%
Multiple	2	2	100%

DISCUSSION

In our study of fifty cases of typhoid ileal perforation, we found that typhoid perforation occurs in both sexes & in our study males predominated. Perforation occurred in all age groups mainly third and fourth decade. Youngest patient in our study was 15 years & oldest was 72 years. Findings were similar to other studies, male predominance may be related to increased risk of exposure to infection. In all cases perforation was diagnosed either clinically or radiologically. Typhoid etiology was proved retrospectively by blood culture or widal test or histopathological examination.

In all cases duration of disease and clinical features were recorded. Important investigations done were X ray chest erect for diagnosing pneumoperitoneum & blood culture, widal reaction and HPE for evidence of typhoid etiology. Positive blood cultures seen mainly in first week of infection, as the typhoid perforation occur in second to third week the blood cultures will be usually negative. Moreover blood cultures are affected by prior antibiotic treatment in early course of disease. Widal test was positive in more cases than blood culture in our study. Widal reaction was positive in

66%, blood culture positive in 32% and histopathological examination suggestive in 60% cases.

Ileal perforation leads to contamination of peritoneal cavity with intestinal secretions containing bacterial flora. This initiates catastrophic events leading to septicemia with multi organ failure which until stopped can be detrimental to the life of patient. The management of ileal perforation was controversial, HUCKSTEP recommended conservative approach whereas HOOK GUERRANS advocated operative approach. At laparotomy ileal perforations were rarely found to be sealed or covered up with omentum. So if the patient is fit for laparotomy, surgical management is always better than conservative approach.

In our study we found that in case of single uncomplicated ileal perforation simple closure suffices. In case of multiple perforations resection of affected segment and end to end anastomosis is required. But in those who are not fit for laparotomy, can be managed by bilateral flank drain. Majority of perforations were limited to distal 25cm segment of ileum in our study. Multiple perforations carry poor prognosis comparing to solitary perforation.

Management of post operative complication is another important aspect. Majority had toxemia & sepsis (40%) in our study. Other complications were wound dehiscence, paralytic ileus, respiratory, fecal fistula etc. Two cases presented with intestinal obstruction as delayed complication. Incidence of complications in our study was comparable to other series like Mock C.N(1992), Kuruvilla(1978), Chauhan(1982) and Mulligan(1972).

Perforations close to ileocaecal junction had higher mortality. Perforations found within 25cm from ileocaecal junction had 29.63% mortality, whereas 30% mortality observed in those perforations found beyond 25cm. Similarly multiple perforations carried poor prognosis in comparison with solitary perforations.

Mortality rate increased with increase in perforation-surgery interval, in our study 8.33% mortality seen when interval was <24 hours, 16.67% when interval was 24-48 hours whereas 71.43% when interval was >96 hours.

Electrolyte imbalance especially Hypokalemia carries poor prognosis. Mortality of 57.14% was seen in those with

hypokalemia comparing to those with normal potassium levels (16.67%).

Mortality was found to be higher in those who presented late in their disease course. In those presented in first week of illness mortality was 12.25%, 15% in those presented in second week of illness whereas it is 60% in those presented late (> 2weeks).

Resection and anastomosis carried higher death rate (36.36%) and simple closure of perforation carried 22.85% mortality. Flank drainage had highest mortality (50%).

In our study Toxemia was most common cause of death accounting 64.29% mortality. Circulatory failure and respiratory complications were major cause of death in remaining patients.

SUMMARY

In our study of fifty cases of Typhoid ileal perforation, 33 cases were males (66%) & 17 cases were females (34%). 56 % cases were belonged to third and fourth decade. All cases were subjected for Widal test which was positive in 33 cases (66%) & Blood culture was positive for S.typhi in 16 cases (32%). In all cases, HPE of ulcer done and it was positive in 30 cases (60%). 45 cases presented during first two weeks of disease course and appear to have good prognosis with mortality of 24.44%.

46 cases underwent operative procedures, among which 35 cases (70%) were treated with simple closure of perforation. 11 cases were treated with Resection and end to end anastomosis with mortality of 36.36%. In 4 cases blind flank drainage was done who were unfit for laparotomy with mortality rate of 50%.

27 cases were having perforation in distal ileum 25 cm from ileocaecal junction. 20 cases were having perforation in ileum 25-50 cm from ileocaecal junction. Perforation site beyond 50 cm from ileoceacal junction were having good prognosis.

Sepsis was the most common complication (40%) in our studies. 20% cases developed post operative paralytic ileus & 14% developed respiratory complications. All patients were followed up for 6 months, 2 patients developed intestinal obstruction who were treated conservatively.

Those with multiple perforations had higher mortality than single perforation. Similarly electrolyte imbalance mainly hypokalemia carried poor prognosis.

Out of fifty cases 14 patients succumb to death, overall mortality was 28%. There was sharp increase in mortality with increase in duration between onset of perforation and surgery. Mortality rate was 8.33% for those with perforation-surgery interval 24 hours where as mortality rate was 71.43% for those with perforation-surgery interval above 96 hours. Most common cause of death in our study was Toxemia (64.29%).

CONCLUSION

1. Typhoid ileal perforation occurs more commonly in males than females, more common in 2-4th decade.
2. Early surgical intervention carries good prognosis.
3. Patients with ileal perforation during the first two weeks of typhoid illness appear to have a better prognosis.
4. Blind drainage may help in those patients, who are not fit for major surgery.
5. Resection is necessary if there are multiple perforations and has equally good prognosis is simple closure.
6. There is a close association of low potassium with the prognosis. Those having hypokalemia carry poor prognosis.

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PROFORMA

A STUDY OF TYPHOID ILEAL PERFORATION

NAME : DATE OF ADMISSION :
AGE/SEX : DATE OF SURGERY :
IP NO : DATE OF DISCHARGE :
ADDRESS :

DURATION OF SYMPTOMS :
TIME INTERVAL BETWEEN
ONSET OF ILLNESS AND PERFORATION :
TIME INTERVAL BETWEEN PERFORATION
AND OPERATIVE INTERVENTION :

SYMPTOMS : FEVER
HEADACHE
VOMITING
ABDOMINAL PAIN
ABDOMINAL DISTENSION
CONSTIPATION
DIARRHOEA
URINARY DISTURBANCES

SIGNS : PALLOR / DEHYDRATION
ABDOMINAL DISTENSION/TENDERNESS
OBLITERATION OF LIVER DULLNESS
GUARDING / RIGIDITY / FREE FLUID
BOWEL SOUNDS

VITALS : PULSE RATE
BLOOD PRESSURE
RESPIRATORY RATE
TEMPERATURE
URINARY OUTPUT

LABORATORY INVESTIGATIONS:

HEMOGLOBIN
RANDOM BLOOD SUGAR

BLOOD UREA
SERUM CREATININE
SERUM ELECTROLYTES- Na, k, Cl, HCO3
URINE ANALYSIS
WIDAL TEST
BLOOD CULTURE

RADIOLOGICAL INVESTIGATIONS:
X-RAY ABDOMEN ERECT
X-CHEST PA VIEW
ULTRASOUND/ CT ABDOMEN

OPERATIVE FINDINGS :
OPERATIVE PROCEDURE :
POST OPERATIVE COMPLICATIONS:
HPE REPORT :
FOLLOW UP :

ASTER CHART

S.No.	Name	Age	Sex	IP No.	Site of Perforation from ileocecal junction (cms)	Associated lesions	Procedure	Widal Test	Blood culture	HPE	Outcome	Complications
1	Perumal	41	M	52189	30		Simple closure	+	+	+	Death	ST
2	Xavier	32	M	7850	15		Simple closure	+	-	+	Survive	-
3	Saraswathi	14	F	52180	35		Resection anastomosis	-	-	-	Death	ST
4	Ganesan	43	M	67731	20		Simple closure	+	+	-	Survive	-
5	Asha	32	F	19851	15		Simple closure	-	-	+	Survive	P
6	Chellapandi	51	M	61218	20		Resection anastomosis	+	+	-	Survive	A
7	Balamurugan	44	M	89516	15		Flank drain	+	-	+	Survive	ST
8	Santhanam	32	M	42154	15		Simple closure	-	-	+	Death	SWT
9	Chinnammal	22	F	98776	15	Ileocaecal strictue	Resection anastomosis	+	-	+	Survive	A
10	Chitra	25	F	1854	55		Simple closure	+	-	-	Survive	P
11	Durai pandi	53	M	48912	20		Resection anastomosis	-	+	-	Survive	F
12	Ganesan	35	M	81214	40		Simple closure	+	-	+	Survive	R
13	Vasudevan	47	M	7581	45		Flank drain	-	+	+	Death	ST
14	Latha	33	F	48321	15		Simple closure	+	-	-	Survive	-
15	Mohan	56	M	32451	35		Simple closure	-	-	+	Death	ST
16	Anitha	26	F	69821	15		Simple closure	+	-	+	Survive	-
17	Alagar	31	M	58710	30		Simple closure	+	+	-	Survive	P
18	Pandi	61	M	78004	15		Resection anastomosis	-	-	-	Survive	F
19	Ramya	37	F	82510	20		Simple closure	+	-	+	Death	P
20	Vellaisamy	31	M	7851	35		Simple closure	-	+	+	Survive	-
21	Kalyani	23	F	64501	40		Resection anastomosis	+	-	+	Death	ST
22	Gowtham	55	M	55231	30		Simple closure	+	-	+	Survive	S
23	Chinnammal	32	F	8942	35		Simple closure	+	+	-	Survive	-
24	Satheeshkumar	62	M	98092	20		Resection anastomosis	-	-	-	Death	ST
25	Rakkkammal	39	F	1891	15		Simple closure	+	-	+	Survive	I
26	Kannan	63	M	45214	15		Simple closure	+	-	+	Survive	S
27	Muthukrishnan	48	M	43211	45		Simple closure	+	-	+	Survive	ST
28	Maheshwari	42	F	51251	20		Simple closure	+	+	+	Survive	S
29	Suresh	22	M	40818	30		Simple closure	+	-	-	Survive	P
30	Tamilmuthu	50	M	88618	45		Simple closure	-	-	-	Death	SWTF

31	Kandasamy	31	M	71251	35		Resection anastomosis	+	+	+	Survive	P
32	Senthilkumar	28	M	42163	20		Simple closure	-	+	-	Survive	S
33	Pratheepa	24	F	31818	20	Ileocaecal strictue	Resection anastomosis	+	-	+	Death	R
34	Kathiravan	37	M	98910	55		Flank drain	+	-	+	Survive	ST
35	Poongodi	33	F	87725	15		Simple closure	-	-	-	Survive	P
36	Bose	39	M	35441	20		Simple closure	+	-	+	Survive	P
37	Vasudevan	19	M	4089	15		Simple closure	-	-	-	Survive	-
38	Jeyapandi	54	M	92441	15		Simple closure	+	-	+	Death	R
39	Bharathiraja	26	M	450619	20		Simple closure	+	-	-	Survive	ST
40	Priya	25	F	42143	45		Simple closure	-	+	-	Survive	-
41	Pandi	36	M	4489	30		Resection anastomosis	+	-	+	Survive	SWTI
42	Lakshmi	51	F	9180	15		Simple closure	-	-	+	Survive	P
43	Pappammal	44	F	94231	20		Simple closure	+	-	-	Death	ST
44	Karuppasamy	27	M	54218	35		Simple closure	+	+	+	Survive	R
45	Mookkan	17	M	4877	15	Ileocaecal strictue	Resection anastomosis	-	-	+	Survive	R
46	Natarajan	33	M	70218	45		Flank drain	-	-	-	Death	ST
47	Durai pandi	24	M	94781	55		Simple closure	+	+	+	Survive	P
48	Gomathi	36	F	34815	20		Simple closure	+	-	-	Survive	R
49	Mahalingam	72	M	52182	20		Simple closure	+	+	+	Survive	R
50	Aruna	43	F	87810	45		Simple closure	+	+	+	Death	ST

KEY TO MASTER CHART

T - TOXEMIA

S - SEPSIS

W - WOUND DEHISCENCE

P - PARALYTIC ILEUS

R - RESPIRATORY COMPLICATIONS

F - FEACAL FISTULA

I - INTESTINAL OBSTRUCTION